UNITED STATES DISTRICT COURT
SOUTHERN DISTRICT OF NEW YORK

IN RE: Acetaminophen – ASD-ADHD Products Liability Litigation **This Document Related To: All Cases**	**22md3043 (DLC)**

REPLY MEMORANDUM IN SUPPORT OF PLAINTIFFS' RULE 702 MOTION TO EXCLUDE DR. MITCHELL R. MCGILL

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In their opposition brief ("Opp.," Dkt. 1255), Defendants spill much ink attempting to shoehorn what Dr. McGill did into some kind of articulable and defensible methodology. In the process, Defendants also misconstrue the applicable standard for the admissibility of rebuttal experts. Their efforts are too little and too late. Without a reliable methodology, Dr. McGill's opinions do not pass muster under Rule 702 and the case law construing it. Moreover, and unsurprisingly given that Dr. McGill's opinions lack a reliable methodology, those opinions are independently inadmissible because they reflect his fundamental misunderstanding and misapplication of the relevant scientific literature.

I. Defendants Get the Applicable *Daubert* Standard Wrong.

In their zeal to insulate their experts from appropriate criticism and disqualification, Defendants misstate the standard that applies to the admissibility of their experts' opinions. Their watered-down version of Rule 702 does not apply to Dr. McGill's affirmative opinions. And even if one were to consider Dr. McGill a pure rebuttal witness—and he is not—he was still required to offer reliable testimony grounded in science.[1] His opinions fail that requirement.

II. Dr. McGill's Purported "Methodology" Is No Methodology At All.

Defendants apparently cannot decide whether the methodology that Dr. McGill supposedly employed was the "scientific method" (Opp. at 8) or a "literature review" (Opp. at 7); they toggle back and forth between the two. Their prevarication is of no moment, however, because Dr. McGill did neither, at least not in any meaningful sense of either phrase. Where an expert's approach "amounts to no methodology at all," exclusion of that expert's opinion is appropriate. *Alto v. Sun Pharms. Indus., Inc.*, No. 1:19-cv-09758-GHW, 2021 WL 4803582, at *7 (S.D.N.Y. Oct. 13, 2021). That is the situation here.

[1] Dr. McGill offers affirmative opinions concerning GSH and AM404 levels in the brain and formation of NAPQI, among other things.

A. The "Scientific Method," Which Dr. McGill Claimed To Have Employed, Is Inapposite.

Defendants alternately chide Plaintiffs for invoking Dr. McGill's testimony describing his method as "the scientific method," but then double down on that testimony. *Compare* Opp. at 8 (criticizing Plaintiffs for relying on "semantics") *with id.* ("[I]t is clear from Dr. McGill's report and deposition that his methodology followed the scientific process, including observation, hypothesis generation, prediction, testing and evaluation of the evidence."). The reason for this prevarication is obvious. Dr. McGill identified no methodology in his report, as Defendants do not dispute. *See* McGill Dep. Tr. at 42:25-43:8, Dkt. 1151-2 ("I didn't describe the methodology in detail within the report. I think it's evident from the structure of the report."). Although it is clear enough that he conducted *some* kind of literature review, the phrase "literature review" is not a methodology. There are distinct and well-established ways of conducting a scientifically reliable review, but Dr. McGill does not mention them (or even show awareness they exist). Dr. McGill's "scientific method" testimony is *all* he has to explain what he did, so Defendants are forced to defend it as a meaningful description of a literature review.

It is not. In the first place, for an expert in the biological sciences to declare his methodology to be the "scientific method" is meaningless, the equivalent of a damages expert claiming "math" as his method. In any event, even the general description of "the scientific method" that Dr. McGill himself articulated assumes experimentation and replication of results, which are part of the familiar understanding of the term, as this Court has noted. *See Fed. Hous. Fin. Agency v. Nomura Holding Am. Inc.*, No. 11cv6201 (DLC), 2015 WL 539489, at *5 (S.D.N.Y. Feb. 10, 2015) (defining the scientific method as "'the process of generating hypotheses and *testing them through experimentation*, publication, and *replication*.'") (emphases added) (quoting Black's Law Dictionary 1547 (10th ed. 2014)). Dr. McGill, however, admitted he conducted no

experiments. *See* McGill. Tr. at 46:14-20, Dkt. 1151-2. And, as discussed further below, his work cannot be replicated because he neither disclosed nor even remembers exactly how he came up with the literature he reviewed. Most importantly, reference to "the scientific method" in general tells the Court virtually nothing about the manner in which Dr. McGill identified relevant literature and analyzed it. Defendants dispute *none* of this.

B. Dr. McGill's "Literature Review" Was Unreliable.

Of course, there are specific methodologies that can be employed to perform a proper literature review—the assessments conducted by Plaintiffs' own experts, like Dr. Stan Louie, are examples. *See, e.g.*, Louie Rep. ¶17, Dkt. 1151-4 ("[Medical literature evaluation] is an especially important component in the drug research and development process, where existing studies and data are used to inform the development of new drugs and the evaluation of drug safety. MLE . . . . principles include ability to detect bias that may confound the findings when evaluating the relative strengths and weaknesses of a particular study."). Defendants oddly describe Plaintiffs' recognition of this fact as a concession, but that it is p*ossible* to conduct a reliable literature review does not mean Dr. McGill actually *did* so.

As a threshold matter, a scientifically valid systematic literature review "seek[s] to collate evidence that fits pre-specified eligibility criteria in order to answer a specific research question. [It] aim[s] to minimize bias using explicit, systematic methods documented in advance with a protocol." Ex. 1, Cumpston M., *Cochrane Handbook for Systematic Reviews of Interventions*, Chapter I: Introduction. Cochrane, 2023. A "systematic review methodology" requires a "highly structured, transparent and reproducible methodology." *Id.* The Cochrane Systematic Review provides one possible framework for performing a methodologically-sound literature review. A Medical Literature Evaluation, the type of analysis performed by Dr. Louie and a common

approach in pharmacology (Louie Rep. ¶17, Dkt. 1151-4), is another approach with similarly stringent requirements.[2] Whatever the nuances of different types of literature review, any reliable one must "accurately document all of the steps and judgments in the [systematic review or] SR process using clear language that is understandable to users and stakeholders. A report should provide enough detail that a knowledgeable reader could *reproduce* the SR." Ex. 3, *Institute of Medicine (US) Committee on Standards for Systematic Reviews of Comparative Effectiveness Research*, Ch. 5: Standards for Reporting Systematic Reviews; Eden J., 2011 (emphasis added). Dr. McGill did not adhere to these bedrock principles.

As a result, Dr. McGill's testimony faces two insurmountable hurdles. First, because he failed to provide and cannot reproduce his review protocol, including his search terms, whatever Dr. McGill purported to do remains untestable. That means his method is *per se* unreliable. *See Daniels-Feasel v. Forest Pharms., Inc.*, No. 17 CV 4188-LTS0JLC, 2021 WL 4037820, at *21 (S.D.N.Y. Sept. 3, 2021). Second, Dr. McGill artificially narrowed the scope of his literature review to justify omitting critical data. This independently invalidates his process.[3]

(i) Dr. McGill's Search Terms Remain Unknown and His Process Cannot Be Replicated

Dr. McGill's report, his testimony, and Defendants' opposition brief confirm that he did not record the search terms that he used to identify the scientific literature that he considered. Defendants' explanation is telling: "Although he did not list those terms in his report, he explained

[2] *See* Ex. 2, Christopher S. Wisniewski, Emily P. Jones, Erin R. Weeda, Nicole A. Pilch, Mary Frances Picone, "Chapter 10: Medical Literature Evaluation and Biostatistics," in *Clinical Pharmacy Education, Practice and Research* 143-162 (Dixon Thomas ed., Elsevier 2019) (summarizing "specific components" of an MLE).

[3] Dr. McGill also failed to weight or grade the scientific literature that he considered. Defendants call out vague references in his testimony where he claimed in nebulous terms that he "assessed the strength of the data based on common considerations and science" (McGill Dep. Tr. at 66:16-18, Dkt. 1151-2) and that he supposedly gave more weight to studies with large doses of acetaminophen. Opp. at 15. That does not cut it. Dr. McGill neither described nor (so far as one can tell) applied a weighting methodology—or explained how he applied whatever his approach was to particular studies—so as to allow a sensible determination of whether his weighting was appropriate.

at his deposition that they included terms *such as* 'acetaminophen and brain' and 'brain and CYP2E1 and fetal.'" (Opp. at 9; emphasis added.) It is meaningless that Dr. McGill belatedly identified the "types of terms" (Opp. at 9 n.1) he used—note that he did not even purport to give examples of terms he *actually did* use—since merely knowing the "types of terms" Dr. McGill used does not enable Plaintiffs to replicate Dr. McGill's process. Without actually knowing what search terms Dr. McGill actually did use, one cannot test reliability of his search or determine whether Dr. McGill actually confronted all of the relevant literature (which it appears he did not). Defendants assert that the "Materials Referenced List" (Opp. at 10) annexed to Dr. McGill's report resolves these deficiencies, but that is not so, since by definition that list does not identify the studies that Dr. McGill ultimately declined to consider (or failed to even locate). Defendants cannot overcome this fundamental failure.[4]

Further, Dr. McGill's testimony and Defendants' opposition brief reveal that Dr. McGill cannot even identify with certainty the *database* that he purported to search. As Defendants put it, "Dr. McGill explained in his report and deposition [that] he not only reviewed the literature cited by plaintiffs' experts, but also found additional articles relevant to his opinions by searching Medline *or* PubMed with 'Boolean search terms.'" Opp. at 9 (emphasis added). Medline and PubMed are different databases in which different literature and studies reside,[5] meaning that

[4] Defendants cite a handful of cases outside this circuit for the proposition that exclusion of an expert's opinion is not necessary simply because the expert could not list "every search term." Opp. at 8. Besides not being binding on this Court, those decisions are distinguishable. The decision in *Smith v. Pfizer Inc.*, 714 F. Supp. 2d 845, 850 (M.D. Tenn. 2010), for example, concerned not a dispute about identifying an expert's search terms but rather a disagreement over which terms were appropriate to use and, in any event, the court observed that the expert's "searches were reproducible." And in *Harris Corp. v. Ruckus Wireless, Inc.*, No. 11-CV-618, 2013 WL 12155458, at *3 (M.D. Fla. Jan. 16, 2013), the court was persuaded that, notwithstanding the expert's inability to recall the search terms from memory, the expert's methods could be recreated, which cannot be done here. Finally, *Moonbug Ent. Ltd. v. BabyBus (Fujian) Network Tech. Co.*, No. 21-cv-06536, 2023 WL 4108838, at *3 (N.D. Cal. June 21, 2023) involved "alternate methodologies or search terms" and *not* whether the methodology used was capable of replication.

[5] For example, although PubMed includes literature also available via Medline, the reverse is not necessarily true. *See MEDLINE, PubMed, and PMC (PubMed Central): How are they different?*, National Library of Medicine, *available at* https://www.nlm.nih.gov/bsd/difference.html.

whether he searched one or the other actually matters. Dr. McGill's approach to searches was so scattershot that even *he* is not sure or cannot recall exactly what he did, which is the definition of an unreproducible—and therefore scientifically invalid—approach.

(ii) Dr. McGill Cherry-Picked The Literature

Defendants attempt to explain away the deficiencies inherent in Dr. McGill's process by characterizing important literature as "outside the scope of [his] opinions." Opp. at 12 (lowercase lettering added). Indeed, Defendants argue that five of the studies that Plaintiffs "complain Dr. McGill did not address pertain[] to neurobehavioral outcomes, a topic on which he is not offering opinions." *Id.* Defendants are simply wrong: The studies Plaintiffs flagged, though they do contain behavioral assessments, do not contain *only* behavioral assessments.

Rather, according to the authors of four of these studies, neurobehavioral outcomes are understood to arise from oxidative stress caused by NAPQI. Consider the following studies that Dr. McGill ignored supposedly because of their attention to animal behavior:

- Baker 2023 reported perturbations in animal neurobehavioral outcomes alongside changes in the expression of genes implicated in oxidative stress caused by NAPQI (discussed further below). *See* Dkt. 1151-7.

- Hay-Schmidt 2017 reported that APAP exposure decreased the number of neurons *(i.e.*, caused cell death as a result of oxidative stress) in regions of the brain known to be affected by ASD and ADHD. This reduction in neuronal populations highlights the end result of oxidative stress caused by NAPQI. *See* Dkt. 1151-10.

- Philippot 2022 not only assessed behavioral outcomes, but also conducted in-depth biochemical and molecular analyses, focusing on the brain's response to APAP exposure. This study evaluated oxidative stress markers through mRNA levels of genes such as Nrf2 and Keap1, assessed protein levels crucial for neuronal growth and synaptic strength, and used immunohistochemistry to evaluate changes in synaptic density, which are all critical indicators of the neurotoxic mechanism of APAP. *See* Dkt. 1151-13.

- Saeedan 2018 examined biochemical and histopathological changes indicative of oxidative stress and inflammation. Specifically, the researchers found marked changes in inflammatory and oxidative stress markers (*e.g.*, TBARS levels) across various acetaminophen treatment groups. *See* Dkt. 1151-15.

Whatever reason Dr. McGill ignored these studies, it is not because they do not bear on the biological mechanisms he claimed to be evaluating. The natural inference is that they were excluded because their findings do not accord with Defendants' preferred litigation outcome. That is the definition of cherry-picking.

In any event, Dr. McGill's wholesale dismissal of any study focused on neurobehavioral outcomes is scientifically unjustified. The literature, including the literature discussed above, shows that neurobehavioral outcomes are related to the mechanistic/metabolic changes that Plaintiffs argue APAP causes. In addition to the associations revealed in the studies noted above, Rigobello 2021 conducted *both* animal behavioral assessments *and* GSH/metabolic assessments and found abnormal neurobehavioral outcomes (*see* Rigobello 2021 at Table 1, Dkt. 1151-19) that correlated with GSH/metabolic changes (*id.* at Table 2). This should have at least put Dr. McGill on notice not only that this particular study is relevant (indeed critical) to evaluate, but also that he should at least *consider* neurobehavioral outcomes and other data (*e.g.*, gene expression) that have been correlated with GSH/metabolic changes. Instead, Dr. McGill uses Rigobello 2021 as a red herring, focusing only on its discussion of GSH levels (on which, more below) and ignoring the balance of the data that does not favor Defendants.

Defendants justify Dr. McGill's disregard of Baker 2023 because of the way in which it relied on gene expression in its findings. *See* Opp. at 13:

> "[Baker 2023] also measured changes in gene expression related to GSH and cytochrome p450, but did not measure those chemicals directly. Dr. McGill is not a geneticist and has never sought to analyze genetic issues. Instead, he opines on measured levels of GSH and CYP2E1 as well as other neurochemicals. Neither plaintiffs nor Baker 2023 addresses how the purported gene expression changes found in the study actually affect levels of GSH or the potential for NAPQI formation, and the article does not contradict any of the data set forth in Dr. McGill's report."

Id. Yet Dr. McGill did not limit himself to data regarding "measured levels of GSH and CYP2E1," as Defendants would have it. Even though he is "not a geneticist," he relied on mRNA data (that is, gene expression) numerous times in his report with respect to, *inter alia*, the Human Protein Atlas and the Allen Institute for Brain Sciences Atlas of the Developing Human Brain. McGill Rep. ¶41, Dkt. 1151-1. Dr. McGill also cited at least *six* published studies of human and rodent brains that he interprets as showing low amounts of CYP2E1 mRNA in the brain. *Id.* at ¶¶42-43 (discussing Boutelet-Bochan et al. (1997), McFayden et al. (1998), Brzezinski et al. (1999), Choudhary et al. (2005), Duthiel et al. (2009), and Tindberg and Ingelman-Sundberg (1996)). Dr. McGill cannot rely on gene expression data when he thinks it supports his position but ignore gene expression data that undermines it.

In short, Defendants cannot explain away Dr. McGill's cherry-picking and disregard of obviously relevant studies on the ground that neurobehavioral outcomes or gene expression data correlated with GSH/metabolic changes—the focus of his report—are somehow outside the scope of his review. These flaws are disqualifying.

III. Dr. McGill's Application Of The Science Is Unreliable.

The unsoundness of Dr. McGill's methodology is borne out in the unreliability of his opinions, which suffer from a fundamental misapprehension of the relevant scientific literature. As described further in Plaintiffs' opening brief, Dr. McGill flatly misread certain critical literature. Pl. Br. at 21, Dkt. 1150. His assessment of Klein 2020 narrowly focuses on the study's failure to find glutathione reduction in parts of the brain while ignoring the ultimate conclusion that the studied rats displayed neurobehavioral alterations "relevant to neurodevelopmental disorders such as ASD and ADHD, as described for PAR-exposed children during pregnancy." Klein 2020 at 7; *see also id.*, Table 1. There, rat pups exhibited "reduced emotionality and

increased responsivity of the dopaminergic system in both male and female pups and also an increased locomotion in females exposed to the drug during pregnancy." *Id.*

Dr. McGill's myopic focus on GSH levels betrays his misunderstanding of the neurobiological processes at issue. The primary biological mechanism he purported to evaluate is oxidative stress caused by an excessive amount of NAPQI in the fetal brain. That can occur even if GSH levels decrease minimally, because GSH levels temporarily increase in order to neutralize the NAPQI.[6] In fact, Dr. McGill's own published research reflects this reality. McGill 2013 shows that APAP conjugates can form in the liver at subtoxic doses of APAP and that extensive GSH depletion is not necessary for the production of injury. That finding squares neatly with Plaintiffs' proposed biological mechanism theory. Not surprisingly, Defendants do not address this tension in their opposition brief.

Dr. McGill also misreads Koehn 2019 as indicating that acetaminophen and its conjugates may differentially traverse the fetal blood brain barrier, even though the study's authors reached the opposite conclusion. Specifically, the study analyzed the ratios of brain/plasma and cerebral spinal fluid/plasma, with the authors explaining that "[r]atios less than 100% indicate restricted entry of the drug or marker into the brain." *Id.* at 12-13. While in adult rats "the brain/plasma and CSF/plasma ratios decreased by approximately 10%" after administration of APAP, "[i]n fetal animals the opposite effect was observed, with an increase in both ratios to around 100%." *Id.* at 7. Thus, the study found that all forms of acetaminophen traversed the fetal blood brain barrier with virtually no restriction.

Koehn 2019's finding, further, belies Defendants' excuse for Dr. McGill's reliance on

[6] *See* Louie Rebuttal Rep. ¶14, Dkt. 1177-10; Ex. 4, Kim SJ, Lee MY, Kwon DY, Kim SY, Kim YC. *Alteration in metabolism and toxicity of acetaminophen upon repeated administration in rats.* J Pharmacol Sci. 2009 Oct; 111(2):175-81.

irrelevant studies to defend his contention that the blood-brain barrier "partially inhibit[s] or block[s] acetaminophen from reaching the [fetal] brain." Opp. at 19. Recall that Dr. McGill relied on a study about PFAS and bisphenols for that proposition, even though (as Dr. McGill admitted) PFAS and bisphenol molecules are much larger than APAP molecules. Pl. Br. at 25, Dkt. 1150. Defendants do not dispute this or explain why this study would be relevant, other than to claim Dr. McGill had no choice because "there is no direct data on acetaminophen crossing the blood-brain barrier in the embryo or fetus." Opp. at 19 (quotation marks omitted). Koehn 2019 shows that is wrong. Perhaps Defendants mean there is no data from *human test subjects*, but that merely reflects Defendants' repeated demand for the scientifically impossible or ethically improper.

Dr. McGill is also wrong in his assessment of the inducibility of CYP2E1. Chronic acetaminophen usage can significantly induce CYP2E1 enzymatic activity, elevating the production of the toxic metabolite NAPQI. Specifically, research by Posadas et al. (2010) demonstrates a concentration-dependent increase in CYP2E1 activity in cortical neurons following acetaminophen administration, corroborating the metabolic amplification of CYP2E1 in these neurons. *See* Posadas 2010, Figure 1B, Dkt. 1151-14. In fact, the study authors found a *tenfold* surge in CYP2E1 expression due to APAP. *See id.*, Figure 6B. Dr. McGill's analysis glaringly omits this critical finding. The omission perhaps reflects that Dr. McGill's own background is in single-dose studies of APAP rather than analyzing the impact of multiple doses of the drug. This is but one of the several examples Plaintiffs identify where Dr. McGill, without a recognized methodology and ranging into matters for which his expertise has not prepared him, has stumbled into errors that render his work unreliable. *See also* Pl. Br. at 21-26, Dkt. 1150.

CONCLUSION

The Court should exclude the opinions offered by Dr. Mitchell R. McGill, Ph.D.

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Respectfully submitted,

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